'Too-early' initiation of dialysis?

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To the Editor: In 'Diagnosis and salvage of an immature fistula' by Bhimani and Asif,[1] the interrelationship of several issues—the importance of fistulas, the salvage of fistulas, and the movement toward earlier initiation of dialysis—is apparent.

In the case presentation, the patient began dialysis therapy through a percutaneous catheter with a recent serum creatinine level of 5.2 mg per 100 ml. Assuming reasonably typical body weight, GFR (glomerular filtration rate) was $\sim$10–15 ml min^{-1}. Therefore, the initiation of dialysis was probably in the absence of overt uremic symptoms, in accordance with the recent K/DOQI (Kidney Disease Outcomes Quality Initiative) guidelines that stress earlier initiation and GFR as an important metric—first specified at 10.5 ml min^{-1} [2] and later revised to 15 ml min^{-1}.[3]

Unfortunately, the patient suffered complications of percutaneous dialysis—sepsis and malfunction—requiring several catheter replacements.

It is worth stressing that K/DOQI does not propose the GFR metric as a mandate, and especially not as an urgent mandate. Instead, it notes that 'It is difficult to make a recommendation for initiating renal replacement therapy based solely on a specific level of GFR,' and discusses the difficulty, to date, that clinical studies have had in demonstrating the benefit of early dialysis.[3] Importantly, three conditions have been listed that 'may indicate that dialysis is not yet necessary' despite reaching the proposed GFR, including stable body weight, acceptable nutritional indices, and absence of symptoms.[2]

The case presentation suggests the need to specify a fourth restraint to early initiation of dialysis: the absence of acceptable access. The putative benefits of earlier dialysis are certainly incremental; the purported risk of waiting will accrue slowly over time, not all at the instant of 15 ml min^{-1}. The possible risk of deferring dialysis for a short period while waiting for appropriate access must be weighed against the frequent complications of percutaneous catheters. In a risk vs benefit calculation, it might be concluded that placing a catheter in the relatively asymptomatic patient to begin 'early' dialysis is not warranted. Acceptable alternatives include the salvage of an immature fistula, the placement of a first or new fistula, 'bridging' peritoneal dialysis, even placement of a graft. Many fistulas are usable after only 4–6 weeks; salvaged fistulas or peritoneal dialysis catheters even sooner and newer graft material allows immediate cannulation.

Taken a step further, if a fistula irreversibly thromboses shortly after dialysis initiation, it may be reasonable, in some patients, to stop dialysis until a new appropriate access is available, rather than to reflexively continue dialysis, in all patients, through placement of a percutaneous catheter.

One reason for the epidemic of catheter prevalence may be the 'too-early' initiation of dialysis through catheters.

1. Bhimani B, Asif A. Diagnosis and salvage of an immature fistula. *Kidney Int* 2007; **72**: 126–130.
2. National Kidney Foundation. *NKF-DOQI Clinical Practice Guidelines for Peritoneal Dialysis Adequacy*. National Kidney Foundation: New York, 1997, pp 17–22.
3. K/DOQI clinical practice guidelines and clinical practice recommendations 2006 updates. Hemodialysis adequacy, peritoneal dialysis adequacy, vascular access. *Am J Kidney Dis* 2006; **48**(Suppl 1): S1.

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Response to ' 'Too-early' initiation of dialysis?'

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We concur with Sheldon Hirsch.[1] There should be a high threshold for tunneled hemodialysis catheter insertion. Such devices should only be placed when renal replacement therapy is required.

1. Hirsch S. 'Too-early' initiation of dialysis? *Kidney Int* 2008; **73**: 511.

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Iron sucrose causes greater proteinuria than ferric gluconate in non-dialysis chronic kidney disease

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To the Editor: Agarwal *et al.*[1] demonstrated that a single dose of iron sucrose causes greater proteinuria than ferric gluconate in a crossover trial of 12 patients with stage 3–4 chronic kidney disease. The toxicity of iron preparations on renal tubular epithelial cells is becoming increasingly recognized in experimental settings.[2] Although not addressed in the original design of the trial, using the blood sampling obtained during the first and second phase of the study, a complementary analytical approach would be to examine whether the use of either parenteral iron preparation was associated with a higher incidence of transient elevation in serum creatinine, which would define